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Respiratory effects of trunk inclination in obese and non-obese patients mechanically ventilated for ARDS

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Abstract

Background Adjusting trunk inclination in patients with acute respiratory distress syndrome directly affects physiological variables such as respiratory mechanics and $PaCO_2$ levels. These effects may vary according to the body mass index (BMI) due to differences in lung and chest wall mechanics, highlighting the need for further investigation to clarify the clinical relevance of body position across patient subgroups.

Methods A secondary analysis compared the physiological effects of increasing trunk inclination angles between mechanically ventilated patients with obesity (BMI ≥ 30 kg/m^2) and those without obesity (BMI < 30 kg/m^2).

Results Data from 159 patients collected across seven individual studies were analyzed. The following physiological changes were observed in response to increased trunk inclination: Sixty-five patients with obesity presented a greater decrease in respiratory system compliance (-7.5 [-10; -5] mL/cmH_2O; $p < 0.001$) compared to ninety-four patients without obesity (-3.5 [-7; -0.08] mL/cmH_2O; $p = 0.045$). Lung compliance decreased in obese patients (-7.8 [-12.4; -3.3] mL/cmH_2O; $p < 0.001$), whereas no significant changes were observed in patients without obesity (-5.9 [-14.2; 2.3] mL/cmH_2O; $p = 0.160$). Chest wall compliance decreased by -42.9 [-63.2; -22.6] mL/cmH_2O ($p < 0.001$) in obese patients and by -47.7 [-95.3; -0.15] mL/cmH_2O in non-obese patients ($p = 0.049$). $PaCO_2$ increased in obese patients by 4.6 [1.4; 7.8] mmHg ($p = 0.004$) but not in patients without obesity (2.5 [-0.6; 5.6] ($p = 0.113$). No significant differences were observed in PaO_2/F_IO_2 between phases.

Conclusions Increasing the trunk inclination angle during passive ventilation reduces respiratory system, lung, and chest wall compliance. This effect was more pronounced in obese patients. Moreover, only this population exhibited an increase in $PaCO_2$. We acknowledge the methodological heterogeneity across the included studies, which may have influenced the results. Overall, our results highlight the importance of considering BMI as a significant variable that influences individual physiological responses to changes in bed inclination.

Keywords ARDS, Body position, Supine position, Compliance of respiratory system, Obese

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Background

Trunk inclination in the supine position has generated increasing scientific interest because of its effects on respiratory physiology in patients with acute respiratory distress syndrome (ARDS) [1, 2]. This is because the semi-recumbent position has been associated with decreased respiratory system compliance (C_{RS}) and higher driving pressures compared to the supine-flat position [3, 4]. These positional adjustments were also associated with an impairment in ventilatory efficiency for carbon dioxide removal, suggesting lung overdistension in semi-recumbent position [5]. Conversely, oxygenation may improve in this position, especially in patients with an increase in end-expiratory lung volume (EELV) [6, 7]. Despite these effects, the evidence in patients with ARDS remains limited and heterogeneous, preventing phenotype-specific recommendations.

Obesity is common among patients with ARDS and is characterized by distinct lung mechanical behaviors compared with non-obese individuals. Under passive ventilation in the supine position, obese individuals exhibit higher expiratory esophageal pressures than their non-obese counterparts, resulting in more negative expiratory transpulmonary pressures and a greater propensity for dependent collapse and atelectasis [8, 9]. These baseline physiological differences suggest that obese patients could exhibit a differential respiratory response when the trunk inclination is modified. Indeed, a study examining the transition from a supine-flat to a semi-recumbent position reported a greater reduction in C_{RS} and an increase in arterial carbon dioxide tension ($PaCO_2$) in obese patients compared to non-obese patients [10]. However, the current evidence is primarily derived from a small, single-center study, which limits its external validity.

We hypothesized that in obese patients with ARDS, increasing trunk inclination would accentuate changes in C_{RS} compared to non-obese patients with ARDS. Accordingly, the primary objective of this study was to analyze the effect of increasing the angle of trunk inclination on C_{RS} in ARDS patients with and without obesity. Secondary objectives were to evaluate the effects of trunk inclination on partitioned respiratory mechanics, arterial oxygen partial pressure to inspired oxygen fraction ratio (PaO_2/F_IO_2), and $PaCO_2$.

Methods

A secondary data analysis was conducted based on previously published studies that evaluated the effects of trunk inclination on respiratory variables in patients with ARDS under passive mechanical ventilation. All included studies received approval from their respective local research ethics boards.

The studies included in this analysis were identified through a previously conducted scoping review on the topic [1]. The lead and corresponding authors were contacted directly via email and asked to dichotomize their original datasets according to body mass index (BMI), classifying them into two predefined categories: non-obese (BMI < 30 kg/m^2) and obese (BMI ≥ 30 kg/m^2). After stratification, demographic, clinical, and physiological data were provided for pooled and subgroup analyses. Additionally, data from a study that included only non-obese patients were also included in the analysis [11].

The patients included in the selected studies were adults (≥ 18 years) with ARDS, invasively mechanically ventilated for fewer than seven days, and under deep sedation or neuromuscular blockade. Each study included in the analysis assessed the respiratory effects of increasing the trunk inclination angle by comparing two different postures in the same patient (Additional file eTable 1). The measured effect corresponded to the transition from a baseline supine position (regardless of the initial inclination angle) to a more elevated trunk position.

Physiological changes associated with transitioning patients to a more upright position were evaluated. The effects of trunk inclination were recorded for each patient using the same positive end-expiratory pressure (PEEP) level at each step. The designs and methodological characteristics of the analyzed studies, including population, mechanical ventilation mode, the timing of PEEP setting, the tool used to determine baseline PEEP, body position during PEEP titration, baseline ventilator settings, protocol phases, and data extraction, are provided in the Additional file (eTable 1).

Data extraction

In these studies, the variables of interest were evaluated at sequential time intervals ranging from 10 to 60 min, with measurements consistently recorded at the end of each stage. The data analysis included the following variables: respiratory mechanics, PaO_2/F_IO_2, EELV, $PaCO_2$, ventilatory ratio (VR), and ventilatory inefficiency variables (Bohr dead space (VD/VT), and the phase III slope of the capnogram (SIII) normalized with fraction of expired CO_2 (F_ECO_2) (SnIII)).

Outcomes

Primary outcome: Changes in C_{RS}.

Secondary outcomes: Changes in partitioned respiratory mechanics (lung and chest wall compliance (C_{CW})), PaO_2/F_IO_2, $PaCO_2$, ventilatory ratio (VR), and ventilatory inefficiency data.

Statistical analysis

Continuous variables from each study were recorded as means and standard deviations (SD). For each study, the treatment effect was represented by the difference in the means between the two positions and was presented using a forest plot. A weighted measure of variability was calculated by considering the variances and sample sizes to obtain the combined standard deviation. The standard error (SE) indicates the uncertainty of estimating treatment effects. The weight of each study and the calculation of the overall effect were recorded using the fixed-effects model (common) and the random effects model (random). Confidence intervals (CI): Inverse Variance (IV), fixed; 95% confidence interval for the mean difference. A result was considered statistically significant when the confidence interval did not include zero value. Two-tailed Z-test to assess whether the combined mean difference between positions significantly differs from zero. A $p < 0.05$ was considered statistically significant. No imputation for missing data was conducted. Analyses were carried out using complete-case data only, based on the information reported in the original studies. Statistical analyses were conducted using RStudio version 4.4.1.

Table 1 Baseline characteristics of the analyzed studies

	BMI < 30 kg/m^2 (n = 94)	BMI ≥ 30 kg/m^2 (n = 65)	*P*-value
Demographics and overall patients' characteristics			
Body mass index (kg/m^2) mean (SD)	25.2 (2.8)	35.5 (4)	< 0.001
Age (years) mean (SD)	63 (10)	62 (11)	0.56
Sex male/female (%)	64%/36%	53%/47%	0.197
Days of mechanical ventilation prior to study onset	3 (2)	3 (2)	-
Ventilatory settings and mechanics			
PEEP (cmH_2O) mean (SD)	10.5 (1.2)	12.2 (2.3)	< 0.001
Driving pressure (cmH_2O) mean (SD)	13.9 (1.3)	12.3 (1.5)	< 0.001
C_{RS} (mL/cmH_2O) mean (SD)	34.5 (9)	34 (9)	0.731
Respiratory rate mean (SD)	22.8 (4.8)	20.7 (10.1)	0.122
Gas exchange and ABG parameters			
PaO_2/F_IO_2 (mmHg) mean (SD)	148 (54)	163 (43)	0.053
PaO_2/F_IO_2 ≥ 200 (mmHg) (n)	8	10	-
PaO_2/F_IO_2 ≥ 100 - < 200 (mmHg) (n)	35	31	-
PaO_2/F_IO_2 < 100 (mmHg) (n)	36	15	-
$PaCO_2$ (mmHg) mean (SD)	47 (7.7)	49 (5.6)	0.059
Etiology			
Pulmonary ARDS (%)	91.5%	84.6%	0.275
Extrapulmonary ARDS (%)	8.5%	15.4%	0.175

PEEP: Positive end–expiratory pressure; C_{RS}: Compliance of respiratory system; PaO_2/FIO_2: Arterial oxygen partial pressure to inspired oxygen fraction ratio; $PaCO_2$: Arterial partial pressure of carbon dioxide; ABG: Arterial blood gas

Results

Data from 159 patients collected across seven individual studies were analyzed [3–5, 10–13]. Sixty-five patients were included in the group with BMI ≥ 30 kg/m^2, and 94 patients in the group with BMI < 30 kg/m^2. The mean BMI was 37.7 ± 4 kg/m^2 and 24.8 ± 2.8 kg/m^2 for obese and non-obese patients, respectively. Changes in C_{RS} were recorded for all the patients. Partitioned respiratory mechanics (lung compliance and C_{CW}) were assessed in 99 patients (46 obese and 53 non-obese). PaO_2/F_IO_2 was measured in 114 patients (49 obese and 65 non-obese), and $PaCO_2$ and VR were measured in 97 patients (49 obese and 48 non-obese). One study analyzed the effects of transitioning trunk inclination on EELV [12], and another analyzed the effects on ventilatory efficiency using volumetric capnography [5]. The Bohr dead space and SnIII were evaluated in a subset of 12 obese and 10 non-obese patients. The baseline characteristics of the analyzed studies are shown in Table 1.

In patients with a BMI ≥ 30 kg/m^2, the transition from supine to a more inclined trunk position significantly decreased C_{RS} from 40.2 ± 9.2 to 32.6 ± 8.6 mL/cmH_2O, with a weighted mean difference of − 7.5 [85% CI − 10; − 5] mL/cmH_2O ($p < 0.001$). In patients with a BMI < 30 kg/m^2, the transition from supine to a more inclined trunk position reduced C_{RS} from 37.4 ± 11.2 to 33 ± 10 mL/cmH_2O, corresponding to a weighted mean difference of − 3.5 [95% CI − 7; − 0.1] mL/cmH_2O ($p = 0.045$). In addition, when C_{RS} changes were compared between the two BMI groups, obese patients exhibited a significantly greater reduction than non-obese patients ($p = 0.04$) (Fig. 1; see Table 1A-B in the Additional file).

In patients with a BMI ≥ 30 kg/m^2, the transition from supine to a more inclined trunk position led to a significant decrease in lung compliance, from 54.4 ± 17.1 to 45.8 ± 17.5 mL/cmH_2O, with a weighted mean difference of − 7.8 [95% CI − 12.4; − 3.3] mL/cmH_2O ($p < 0.001$). In contrast, in patients with a BMI < 30 kg/m^2, lung compliance showed no significant decrease, from 50.5 ± 19 to 43.8 ± 17 mL/cmH_2O with a weighted mean difference of − 5.9 [95% CI − 14.2; 2.3] ($p = 0.160$) under the same positional change (Fig. 2A). In patients with a BMI ≥ 30 kg/m^2, transitioning from supine to a more inclined trunk position significantly decreased C_{CW} from 167.8 ± 55.7 to 120.6 ± 34.1 with a weighted mean difference of − 42.9 [95% CI − 63.2; − 22.6] mL/cmH_2O ($p < 0.001$). Similarly, in patients with a BMI < 30 kg/m^2, transitioning from supine to a more inclined trunk position reduced C_{CW} from 173.9 ± 90 to 127.1 ± 49.1 mL/cmH_2O, corresponding to a weighted mean difference − 47.7 [95% CI − 95.3; − 0.15] mL/cmH_2O ($p = 0.049$) (Fig. 2B). In addition, no significant differences were observed in lung and chest wall compliance between the two BMI groups ($p = 0.59$ and $p = 0.76$, respectively).

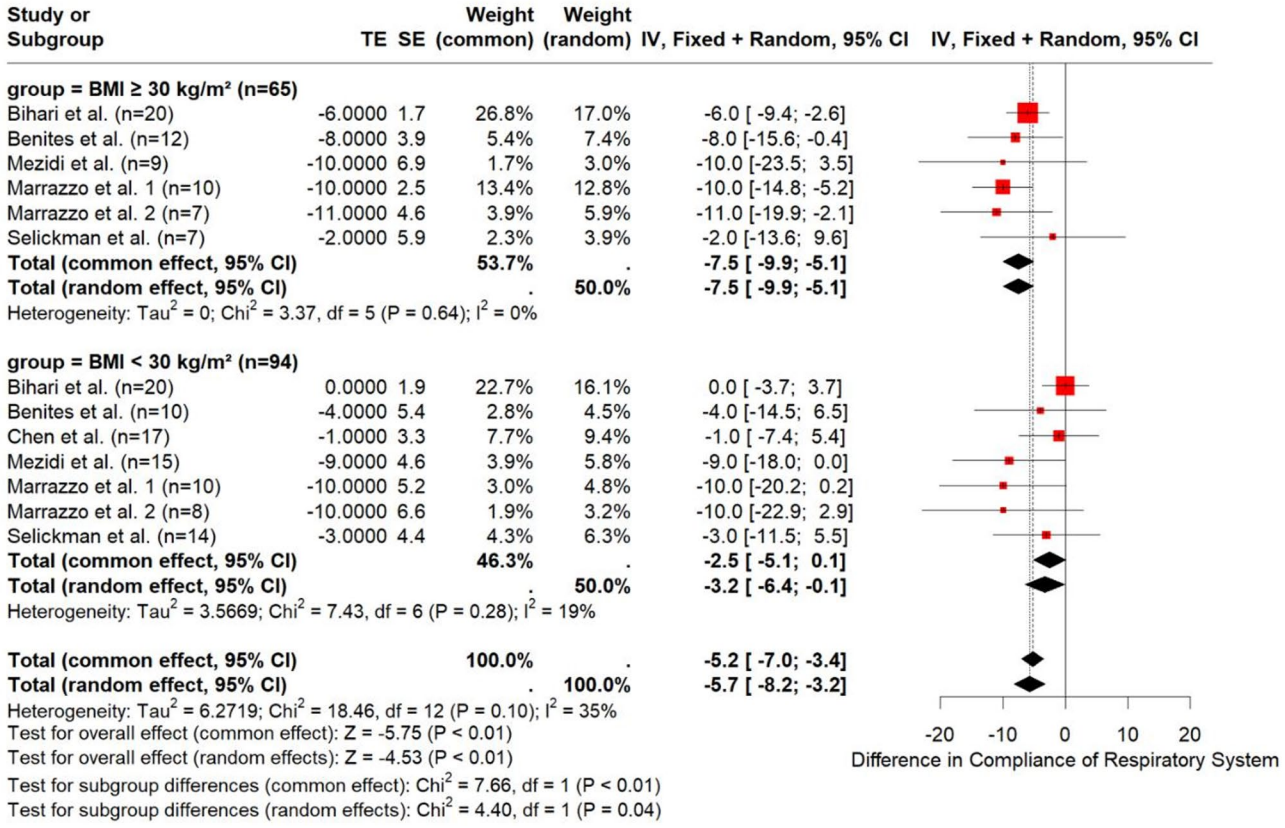
Study or Subgroup
TE SE
Weight (common)
Weight (random)
IV, Fixed + Random, 95% CI
IV, Fixed + Random, 95% CI
group = BMI ≥ 30 kg/m² (n=65)
Bihari et al. (n=20) -6.0000 1.7 26.8% 17.0% -6.0 [-9.4; -2.6]
Benites et al. (n=12) -8.0000 3.9 5.4% 7.4% -8.0 [-15.6; -0.4]
Mezidi et al. (n=9) -10.0000 6.9 1.7% 3.0% -10.0 [-23.5; 3.5]
Marrazzo et al. 1 (n=10) -10.0000 2.5 13.4% 12.8% -10.0 [-14.8; -5.2]
Marrazzo et al. 2 (n=7) -11.0000 4.6 3.9% 5.9% -11.0 [-19.9; -2.1]
Selickman et al. (n=7) -2.0000 5.9 2.3% 3.9% -2.0 [-13.6; 9.6]
Total (common effect, 95% CI) 53.7% -7.5 [-9.9; -5.1]
Total (random effect, 95% CI) 50.0% -7.5 [-9.9; -5.1]
Heterogeneity: Tau² = 0; Chi² = 3.37, df = 5 (P = 0.64); I² = 0%
group = BMI < 30 kg/m² (n=94)
Bihari et al. (n=20) 0.0000 1.9 22.7% 16.1% 0.0 [-3.7; 3.7]
Benites et al. (n=10) -4.0000 5.4 2.8% 4.5% -4.0 [-14.5; 6.5]
Chen et al. (n=17) -1.0000 3.3 7.7% 9.4% -1.0 [-7.4; 5.4]
Mezidi et al. (n=15) -9.0000 4.6 3.9% 5.8% -9.0 [-18.0; 0.0]
Marrazzo et al. 1 (n=10) -10.0000 5.2 3.0% 4.8% -10.0 [-20.2; 0.2]
Marrazzo et al. 2 (n=8) -10.0000 6.6 1.9% 3.2% -10.0 [-22.9; 2.9]
Selickman et al. (n=14) -3.0000 4.4 4.3% 6.3% -3.0 [-11.5; 5.5]
Total (common effect, 95% CI) 46.3% -2.5 [-5.1; 0.1]
Total (random effect, 95% CI) 50.0% -3.2 [-6.4; -0.1]
Heterogeneity: Tau² = 3.5669; Chi² = 7.43, df = 6 (P = 0.28); I² = 19%
Total (common effect, 95% CI) 100.0% -5.2 [-7.0; -3.4]
Total (random effect, 95% CI) 100.0% -5.7 [-8.2; -3.2]
Heterogeneity: Tau² = 6.2719; Chi² = 18.46, df = 12 (P = 0.10); I² = 35%
Test for overall effect (common effect): Z = -5.75 (P < 0.01)
Test for overall effect (random effects): Z = -4.53 (P < 0.01)
Test for subgroup differences (common effect): Chi² = 7.66, df = 1 (P < 0.01)
Test for subgroup differences (random effects): Chi² = 4.40, df = 1 (P = 0.04)
-20 -10 0 10 20
Difference in Compliance of Respiratory System

Fig. 1 Forest Plot of Compliance of Respiratory System in Patients with BMI ≥ 30 kg/m^2 vs. BMI < 30 kg/m^2. Transitioning from supine to a more inclined trunk position. TE (Treatment Effect): Estimated effect for each study, representing the mean difference in Compliance of respiratory system between the obese and non–obese groups. SE (Standard Error): Weight (common/random): IV (Inverse Variance), Fixed; 95% CI, 95% confidence interval (CI) for the estimated effect under a fixed–effects model. Heterogeneity within subgroups: Tau^2: An estimate of variance between studies. Chi^2: Chi–squared statistic to assess heterogeneity. I^2: Percentage of total variation attributable to heterogeneity between studies. A value of 0% indicated low heterogeneity. Each group is represented by a black diamond, indicating the combined effect (meta–analysis) for that subgroup. The endpoints of diamond reflect the 95% confidence interval (CI). The total at the bottom of the group provides a combined result for all studies within that subgroup. Vertical dotted line: This is the weighted average of all included studies. Continuous vertical line at value 0: Null effect

In obese patients, a change in trunk inclination from the supine to a more inclined trunk position resulted in a significant decrease in the ratio between lung elastance and Elastance of respiratory system (E_L/E_{RS}) and a concurrent increase in its counterpart of the ratio between chest wall elastance and elastance of respiratory system (E_{CW}/E_{RS}) (Table 2). In contrast, no significant changes in these variables were observed among the non-obese patients (Table 3; see supplementary Figs. 1 and 2 in the Additional file).

The respiratory mechanics and arterial blood gas results are summarized in Tables 2 (obese patients) and 3 (non-obese patients). Grouped data from the included studies are reported in both tables. Each study-level dataset is presented in the supplementary additional file for detailed reference.

Forest plots of PaO_2/F_IO_2, $PaCO_2$, VR, and ventilatory inefficiency data are presented in the Additional file.

Discussion

The main findings of the present analysis were as follows: (1) a more inclined trunk position led to a reduction in C_{RS} in both obese and non-obese patients; (2) this decrease in C_{RS} was more pronounced in patients with a BMI ≥ 30 kg/m^2 than in those with a BMI < 30 kg/m^2; and (3) only obese patients exhibited a significant increase in $PaCO_2$ following this postural change.

These findings provide comparative insights into the effects of body positioning on respiratory mechanics in obese and nonobese patients. Additionally, these findings suggest that patients with obesity may represent a distinct subgroup characterized by greater susceptibility to changes in respiratory mechanics in response to variations in trunk inclination. Accordingly, particular attention should be paid to the physiological impact of this intervention in this population.

In obese and non-obese patients, alveolar collapse in dependent lung regions is promoted by gravitational forces, which generate superimposed pressure [14]

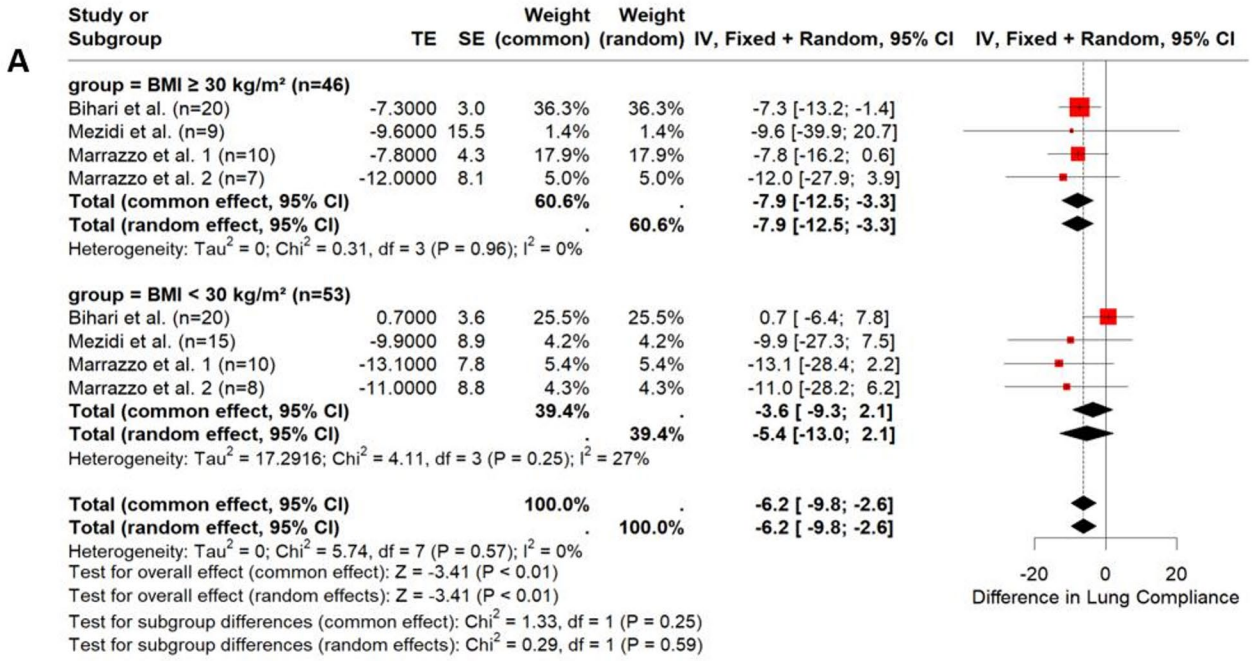
A
Study or Subgroup
TE
SE
Weight (common)
Weight (random)
IV, Fixed + Random, 95% CI
IV, Fixed + Random, 95% CI
group = BMI ≥ 30 kg/m² (n=46)
Bihari et al. (n=20) -7.3000 3.0 36.3% 36.3% -7.3 [-13.2; -1.4]
Mezidi et al. (n=9) -9.6000 15.5 1.4% 1.4% -9.6 [-39.9; 20.7]
Marrazzo et al. 1 (n=10) -7.8000 4.3 17.9% 17.9% -7.8 [-16.2; 0.6]
Marrazzo et al. 2 (n=7) -12.0000 8.1 5.0% 5.0% -12.0 [-27.9; 3.9]
Total (common effect, 95% CI) 60.6% . -7.9 [-12.5; -3.3]
Total (random effect, 95% CI) . 60.6% -7.9 [-12.5; -3.3]
Heterogeneity: Tau² = 0; Chi² = 0.31, df = 3 (P = 0.96); I² = 0%
group = BMI < 30 kg/m² (n=53)
Bihari et al. (n=20) 0.7000 3.6 25.5% 25.5% 0.7 [-6.4; 7.8]
Mezidi et al. (n=15) -9.9000 8.9 4.2% 4.2% -9.9 [-27.3; 7.5]
Marrazzo et al. 1 (n=10) -13.1000 7.8 5.4% 5.4% -13.1 [-28.4; 2.2]
Marrazzo et al. 2 (n=8) -11.0000 8.8 4.3% 4.3% -11.0 [-28.2; 6.2]
Total (common effect, 95% CI) 39.4% . -3.6 [-9.3; 2.1]
Total (random effect, 95% CI) . 39.4% -5.4 [-13.0; 2.1]
Heterogeneity: Tau² = 17.2916; Chi² = 4.11, df = 3 (P = 0.25); I² = 27%
Total (common effect, 95% CI) 100.0% . -6.2 [-9.8; -2.6]
Total (random effect, 95% CI) . 100.0% -6.2 [-9.8; -2.6]
Heterogeneity: Tau² = 0; Chi² = 5.74, df = 7 (P = 0.57); I² = 0%
Test for overall effect (common effect): Z = -3.41 (P < 0.01)
Test for overall effect (random effects): Z = -3.41 (P < 0.01)
Test for subgroup differences (common effect): Chi² = 1.33, df = 1 (P = 0.25)
Test for subgroup differences (random effects): Chi² = 0.29, df = 1 (P = 0.59)
-20
0
20
Difference in Lung Compliance
B
Study or Subgroup
TE
SE
Weight (common)
Weight (random)
IV, Fixed + Random, 95% CI
IV, Fixed + Random, 95% CI
group = BMI ≥ 30 kg/m² (n=46)
Bihari et al. (n=20) -29.3000 10.9 39.2% 23.8% -29.3 [-50.7; -7.9]
Mezidi et al. (n=9) -49.8000 21.9 9.7% 13.5% -49.8 [-92.8; -6.8]
Marrazzo et al. 1 (n=10) -75.0000 29.3 5.4% 9.2% -75.0 [-132.5; -17.5]
Marrazzo et al. 2 (n=7) -55.0000 22.7 9.1% 12.9% -55.0 [-99.5; -10.5]
Total (common effect, 95% CI) 63.4% . -40.0 [-56.9; -23.2]
Total (random effect, 95% CI) . 59.4% -40.2 [-57.1; -23.2]
Heterogeneity: Tau² = 2.4972; Chi² = 3.02, df = 3 (P = 0.39); I² = 1%
group = BMI < 30 kg/m² (n=53)
Bihari et al. (n=20) -5.0000 13.5 25.7% 21.0% -5.0 [-31.4; 21.4]
Mezidi et al. (n=15) -53.4000 27.6 6.1% 10.1% -53.4 [-107.4; 0.6]
Marrazzo et al. 1 (n=10) -58.0000 42.6 2.6% 5.2% -58.0 [-141.4; 25.4]
Marrazzo et al. 2 (n=8) -125.0000 47.1 2.1% 4.4% -125.0 [-217.3; -32.7]
Total (common effect, 95% CI) 36.6% . -23.8 [-46.0; -1.6]
Total (random effect, 95% CI) . 40.6% -48.2 [-96.6; 0.3]
Heterogeneity: Tau² = 1463.7287; Chi² = 8.36, df = 3 (P = 0.04); I² = 64%
Total (common effect, 95% CI) 100.0% . -34.1 [-47.5; -20.7]
Total (random effect, 95% CI) . 100.0% -42.6 [-63.3; -21.8]
Heterogeneity: Tau² = 352.5745; Chi² = 12.69, df = 7 (P = 0.08); I² = 45%
Test for overall effect (common effect): Z = -4.99 (P < 0.01)
Test for overall effect (random effects): Z = -4.02 (P < 0.01)
Test for subgroup differences (common effect): Chi² = 1.31, df = 1 (P = 0.25)
Test for subgroup differences (random effects): Chi² = 0.09, df = 1 (P = 0.76)
-200
-100
0
100
200
Difference in Chest Wall Compliance

Fig. 2 Forest Plot of Lung and Chest Wall Compliance in patients with BMI ≥ 30 kg/m^2 vs. BMI < 30 kg/m^2. Transitioning from supine to a more inclined trunk position. **A**– Lung Compliance. **B**– Chest Wall Compliance. TE (Treatment Effect): Estimated effect for each study, representing the mean difference in Compliance of respiratory system between the obese and non–obese groups. SE (Standard Error): Weight (common/random): IV (Inverse Variance), Fixed; 95% CI, 95% confidence interval (CI) for the estimated effect under a fixed–effects model. Heterogeneity within subgroups: Tau^2: An estimate of variance between studies. Chi^2: Chi–squared statistic to assess heterogeneity. I^2: Percentage of total variation attributable to heterogeneity between studies. A value of 0% indicated low heterogeneity. Each group is represented by a black diamond, indicating the combined effect (meta–analysis) for that subgroup. The endpoints of diamond reflect the 95% confidence interval (CI). The total at the bottom of the group provides a combined result for all studies within that subgroup. Vertical dotted line: This is the weighted average of all included studies. Continuous vertical line at value 0: Null effect

Table 2 Patients with obesity (BMI ≥ 30 kg/m^2). Transition from supine position to a more inclined trunk position

BMI ≥ 30 Kg/m^2	Supine position	A more inclined trunk position	Mean difference [95% CI]	*P* value
PEEP (cmH_2O)	11.6 (1.8)	11.6 (1.8)	0 [−0.5; 0.5]	1
Peak inspiratory pressure (cmH_2O)	30.6 (3.7)	33.4 (3.9)	2.8 [1.5; 4.1]	< 0.001
Driving pressure (cmH_2O)	10.8 (3)	13.5 (3.7)	2.8 [1.9; 3.7]	< 0.001
C_{RS} (mL/cmH_2O)	40.2 (9.2)	32.6 (8.6)	−7.5 [−10; −5]	< 0.001
Lung compliance	54.4 (17.1)	45.8 (17.5)	−7.8 [−12.4; −3.3]	< 0.001
Compliance CW (mL/cmH_2O)	167.8 (55.7)	120.6 (34.1)	−42.9 [−63.2; −22.6]	< 0.001
Lung elastance/elastance RS	0.77 (0.11)	0.71 (0.11)	−0.06 [−0.11; −0.01]	0.011
Elastance CW/elastance RS	0.24 (0.11)	0.29 (0.11)	0.05 [0.00; −0.1]	0.040
Ventilatory ratio (VR)	1.7 (0.36)	1.8 (0.38)	0.1 [−0.04; −0.24]	0.150
$PaCO_2$ (mmHg)	44.9 (5.1)	49 (5.6)	4.6 [1.4; 7.8]	0.004
PaO_2/F_IO_2 (mmHg)	146 (41)	158 (43)	11.9 [−4.3; 28.2]	0.151

Data are expressed as weighted mean differences using a random−effects model and Z−test. PEEP: Positive−end expiratory pressure. C_{RS}: Compliance of respiratory system. C_{CW}: Chest wall. RS: Respiratory system. $PaCO_2$: Arterial partial pressure of carbon dioxide. PaO_2/FIO_2: Arterial oxygen partial pressure to inspired oxygen fraction ratio

Table 3 Patients without obesity (BMI < 30 kg/m^2). Transition from supine position to a more inclined trunk position

BMI < 30 Kg/m^2	Supine position	A more inclined trunk position	Mean difference [95% CI]	*P* value
PEEP (cmH_2O)	10.7 (2.5)	10.7 (2.5)	0 [−0.45; 045]	1
Peak inspiratory pressure (cmH_2O)	30.5 (4.1)	32.7 (4.6)	2.2 [−0.1; 4.5]	0.060
Driving pressure (cmH_2O)	11.8 (3.5)	14.1 (4.8)	2.4 [0.47; 4.44]	0.023
C_{RS} (mL/cmH_2O)	37.4 (11.2)	33 (10)	−3.5 [−7; −0.08]	0.045
Lung compliance	50.5 (19)	43.8 (17)	−5.9 [−14.2; 2.36]	0.160
Compliance CW (mL/cmH_2O)	173.9 (90)	127.1 (49.1)	−47.7 [−95.3; −0.15]	0.049
Lung elastance/elastance RS	0.78 (0.11)	0.75 (0.11)	−0.02 [−0.07; 0.03]	0.566
Elastance CW/elastance RS	0.22 (0.12)	0.27 (0.12)	0.04 [−0.01; −0.09]	0.141
Ventilatory ratio (VR)	1.58 (0.32)	1.63 (0.35)	0.04 [−0.09; 0.17]	0.512
$PaCO_2$ (mmHg)	47 (7.7)	49.3 (8.4)	2.5 [−0.6; 5.6]	0.113
PaO_2/F_IO_2 (mmHg)	142 (48)	142 (54)	−0.76 [−16.4; 14.9]	0.923

Data are expressed as weighted mean differences using a random−effects model and Z−test. PEEP: Positive−end expiratory pressure. C_{RS}: Compliance of respiratory system. C_{CW}: Chest wall. RS: Respiratory system. $PaCO_2$: Arterial partial pressure of carbon dioxide. PaO_2/FIO_2: Arterial oxygen partial pressure to inspired oxygen fraction ratio

and a vertical pleural pressure gradient [15]. These two mechanisms operate simultaneously, contributing to alveolar collapse, and their relative impact is likely to vary according to the severity of lung injury and the thoracic positioning [16]. Superimposed pressure describes the pressure exerted by the weight of the overlying tissue on more dependent pulmonary regions due to gravitational forces. This hydrostatic force, generated by lung weight, is considered a determinant of the pleural pressure gradient [16, 17]. Furthermore, increased body weight and fat around the thorax are key factors that influence superimposed pressure, resulting in greater mechanical compression in dependent regions [14, 15]. The pleural pressure gradient refers to the pressure difference within the pleural space along the vertical axis of the lungs [15]. Although lung morphology and elastance of both the lung and chest wall appear similar between obese and non-obese patients, obesity is characterized by a reduced lung apex-to-base length and an increased sternum-to-vertebral diameter [16]. In supine-flat obese patients, this altered thoracic geometry increases the anteroposterior dimension of the lungs, thereby steepening the gravitational pleural pressure gradient. This anatomical configuration likely contributes to a rightward shift of the pressure-volume relationship, reflecting the greater external mechanical load imposed by the chest wall [16].

In addition, obese patients have increased intra-abdominal adiposity and pressure [18], which may result in greater cephalad displacement of the abdominal contents against the diaphragm when transitioning to a more inclined trunk position through hip flexion compared to reverse Trendelenburg positioning. This, in turn, causes distortion of the thoracic compartment structures, contributing to altered respiratory mechanics and reduced lung compliance [19]. Furthermore, transitioning from the supine position to a more inclined trunk position is expected to increase the vertical height of the lung and accentuate the pleural pressure gradient [20]. Although the volume of the abdominal contents does not change when moving to a semi-recumbent position, the shape of the abdominal cavity is likely distorted. Distortion leads to an increase in intra-abdominal pressure, which is transmitted primarily to the thoracic compartment [21–23]. This positive upward pressure is likely to be concentrated in the dependent juxtadiaphragmatic regions. Consequently, the ventilation distribution in mechanically ventilated obese patients is preferentially directed toward regions with greater compliance, such as non-dependent lung areas. This phenomenon is physiologically translated as a rightward shift of the pressure-volume curve [24], indicating a greater susceptibility to alterations in lung mechanics in obese patients (Fig. 3).

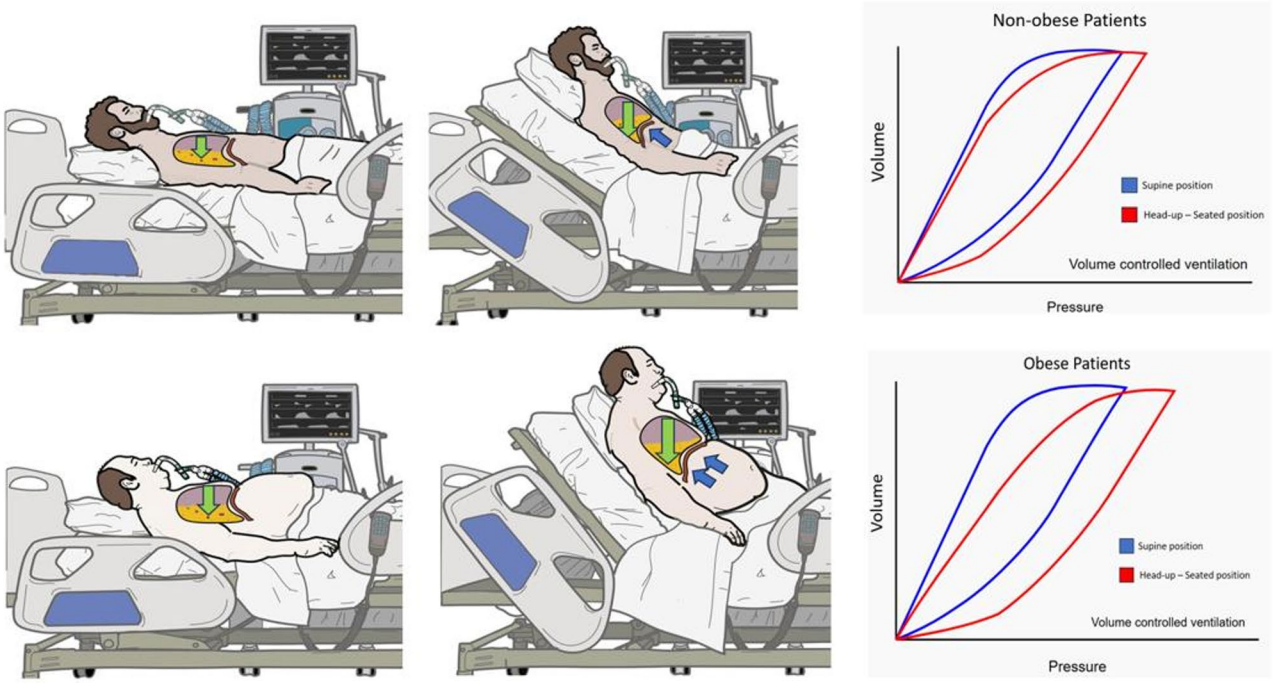

Fig. 3 Transitioning from supine to a more inclined trunk position and its effects on the pressure-volume curve. Top: Non–obese patients under passive ventilation. Bottom: Obese patients under passive ventilation. Changes in trunk inclination generate a rightward shift of the pressure–volume loop (theoretical concept), indicating a decrease in compliance of respiratory system. Green arrows indicate the distances between non–dependent and dependent lung regions, while blue arrows represent the magnitude and direction of the intra–abdominal pressure

In our analysis, the partitional analysis of thoracopulmonary mechanics showed that both obese and non-obese patients with ARDS experienced a reduction in C_{CW} when transitioning from supine to a more inclined trunk position. This effect was accompanied by less variability in data dispersion in obese patients than in non-obese patients, suggesting a more homogeneous response in the obese group. Additionally, obese patients showed a decline in lung compliance after this postural modification. This effect likely reflects a greater collapse in dependent lung regions and overdistension in non-dependent zones, both recognized mechanisms of lung stress and strain [25], particularly when PEEP is not optimally set [26].

We also observed a significant increase in the E_{CW}/E_{RS} ratio in obese patients. This finding suggests that despite increased lung elastance, a greater proportion of the energy delivered by the mechanical ventilator was directed toward the elastic component of the chest wall when the patients adopted a more upright posture. In contrast, non-obese patients did not show a significant redistribution of the elastic load between the lung and chest wall.

To understand the relationship between trunk angle and changes in the pressure–volume curve in obese and non-obese patients, it is essential to consider the results obtained from optimizing PEEP in various body positions. Marrazzo et al. demonstrated that optimized PEEP requires adjustments to be made for each change in the chest position. In the supine-flat position, the optimal PEEP levels are, on average, 5 cmH_2O higher than in the semi-recumbent position. Therefore, when the optimal PEEP is set in the supine-flat position, transitioning to a semi-recumbent position may require reducing the PEEP to avoid alveolar overdistension [26]. Hence, maintaining the same PEEP levels after transitioning from a supine-flat to a semi-recumbent position may contribute to excessive EELV and regional hyperinflation, particularly in patients with low potential for lung recruitment. In this context, it is plausible that trunk elevation produces physiological effects similar to those induced by increasing PEEP in non-recruitable lung regions, leading to increased pulmonary vascular resistance and deleterious effects on the right ventricle [2, 26–28]. Notably, in contrast to previous reports on ventilated obese patients with ARDS [29], the obese cohort in our study received a lower PEEP. This pattern may reflect reduced potential for lung recruitment in obese patients, which could in turn confer greater susceptibility to overdistension [28].

Regarding gas exchange, we observed that $PaCO_2$ levels worsened when patients were transitioned from supine to a more inclined trunk position, with this effect being more pronounced in the obese subgroup. To explain this phenomenon, one study employed volumetric

capnography to evaluate the impact of upward trunk inclination on ventilatory inefficiency [5]. This postural change was accompanied by increased VD_{Bohr}/VT and $PaCO_2$, and the slope of phase III (SIII) of the capnogram, normalized to the fraction of expired CO_2 (F_ECO_2), also increased significantly, suggesting that postural changes in obese individuals may exacerbate ventilation-perfusion mismatch, likely because of overdistension [5]. Collectively, these observations reinforce the link between reduced lung compliance and impaired CO_2 clearance in obesity, indicating that greater trunk inclination may predispose to alveolar overdistension and compromised gas exchange.

It is important to note that this assessment was conducted under pressure-controlled ventilation to assess the effects on ventilatory efficiency [5]. In the same line with other reports, shifting to a more inclined trunk position reduced C_{RS}; however, in this case, as a result of a decrease in tidal volume (VT), consistent with impaired lung mechanics in this posture. Although changes in minute ventilation, particularly those driven by VT, can influence dead-space ventilation [30], the reduction in VT observed with greater trunk inclination is plausibly explained by tidal derecruitment and lung overdistension. This phenomenon likely arises because, at the same inspiratory pressure, upward trunk inclination places patients on a steeper segment of the pressure–volume curve, thereby increasing lung strain. As a result, both the Bohr dead-space and the phase III slope of the capnogram (SnIII) increase, indicating impaired CO_2 clearance efficiency (Additional file).

These results contrast with those of VR, which was unaffected by changes in trunk inclination in both obese and non-obese patients. Although VR is a recognized predictor of clinical outcomes [31], its value as a physiological marker of ventilatory efficiency in ARDS remains unclear [32]. This limitation arises because $PaCO_2$, a core component of VR, can increase through different ventilation–perfusion mismatch mechanisms, including venous admixture and elevated dead space [32]. Consequently, VR correlates closely with Enghoff's VD/VT [31], but poorly with more sensitive indices of ventilatory efficiency, such as CO_2 elimination per breath ($VTCO_{2,br}$) [31], which may explain its minimal response to trunk inclination in our analysis (Additional File).

Regarding the effects of trunk inclination on oxygenation, some studies have examined the impact of increasing the trunk inclination angle on oxygenation and lung volume [2, 6, 7]. Richard et al. and Dellamonica et al. evaluated patients with ARDS after increasing trunk inclination. Richard et al. reported a higher proportion of patients with improved oxygenation, which is closely associated with increases in EELV, whereas Dellamonica et al. observed oxygenation improvement in only a minority of patients, with inconsistent correlations with EELV changes. Despite these differences, both studies consistently demonstrated that increasing trunk inclination reduces C_{RS} [6, 7]. Bouchant et al. showed that when PEEP was titrated in the semi-recumbent position (30°) and remained unchanged during changes in trunk inclination, transitioning patients from the 30° to the supine-flat position (0°) resulted in a decline in oxygenation and EELV [2]. This deterioration likely reflects alveolar derecruitment induced by postural shift without PEEP readjustment [26]. Re-elevation to 30° reversed these effects, suggesting recruitment of previously collapsed lung regions. However, further increases in inclination to 60° and 90° did not yield additional improvements and were associated with a decline in oxygenation and EELV [2]. Therefore, an excessively upright posture without adequate PEEP adjustment may promote overdistension. This effect is believed to result from reduced dorsal atelectasis due to less compression of the dependent lung regions by the cephalad displacement of the abdomen and diaphragm [33] and from a decrease in apical and mid-apical pleural pressures, which increases transpulmonary pressure, the principal determinant of lung distension [34]. Our analysis extended these observations by stratifying patients according to BMI, showing that obese patients experienced greater reductions in C_{RS}, while oxygenation remained essentially unchanged across groups. Taken together, these results highlight the heterogeneity of physiological responses to trunk positioning and emphasize the need for individualized management.

Final comments and clinical implications

This analysis identified obesity as a clinical condition that may predispose patients to respiratory impairment when trunk inclination increases during passive mechanical ventilation. Such impairments likely reflect the combined effects of altered respiratory mechanics and increased abdominal pressure in this population. These findings enhance the external validity of the evidence by demonstrating that the physiological consequences of body positioning are not uniform across all patients, but are influenced by individual phenotypes.

From a practical standpoint, the results underscore the importance of systematically documenting bed inclination and conducting comprehensive respiratory assessments, including gas exchange, lung mechanics, and hemodynamic parameters [2], when evaluating the positioning strategies in patients with ARDS. Understanding that increasing the angle of trunk inclination can alter respiratory mechanics in obese patients should not be interpreted as a requirement for these patients to remain in a supine-flat position. Instead, these findings should encourage a reassessment of the optimal PEEP levels

(e.g., airway opening pressure) in the chosen position, ensuring that adjustments are tailored to each patient's physiological responses.

Incorporating these assessments into daily practice can help clinicians identify patients who may benefit from specific inclination angles and avoid those that could be detrimental. This approach is consistent with the principles of precision medicine, in which mechanical ventilation strategies are tailored to the patient's physiological profile rather than being uniformly applied. It is essential to acknowledge that the heterogeneity of methodologies among the studies included in this analysis may have influenced the results, underscoring the need for prospective, standardized research to confirm these observations. Finally, clinicians should be aware that a supine-flat position, while potentially advantageous in specific respiratory contexts, may be associated with adverse events, such as ventilator-associated pneumonia.

Limitations

The limitations of these analyzed results include several considerations. While the study stratified patients according to BMI status, the analysis included heterogeneous forms of ARDS, including C-ARDS, pulmonary ARDS, and extrapulmonary ARDS. Each of these entities may exhibit distinct pathophysiological profiles, potentially influencing respiratory mechanics and response to postural changes, thereby introducing variability in the observed outcomes. BMI was dichotomized, and a definitive cut-off value could not be established to identify accurately which patients might benefit most from this intervention. Likewise, the heterogeneity of obesity phenotypes (e.g., type III vs. non–type III, android vs. gynoid fat distribution) was not addressed in this study. These subpopulations may exhibit different responses to changes in trunk inclination, depending on the body position in which PEEP was optimized in each study [14].

Although all studies shared the common feature of increasing the trunk inclination angle, they did not start from the same baseline angle or applied an identical degree of thoracic elevation, which may have influenced the magnitude of the observed postural effect. Furthermore, while PEEP settings were maintained constant during the intervention phase in all studies, inter-study variability in baseline PEEP levels could have affected the observed physiological responses to positional changes. In this way, in many of the included studies, the assessment of airway opening pressure was lacking, representing one of the most important limitations of this study. On the other hand, the meta-analytic approach limits the ability to assess variability at the individual patient level, a factor that is relevant for a more accurate assessment of the physiological response to trunk positioning in critically ill patients. Finally, the evaluation time for each posture across included studies did not exceed 60 min, leaving the long-term persistence of these effects unknown.

Conclusions

Increasing the trunk inclination angle during passive ventilation reduces respiratory system, lung, and chest wall compliance. This effect was more pronounced in obese patients. Moreover, only this population exhibited an increase in $PaCO_2$. We acknowledge the methodological heterogeneity across the included studies, which may have influenced the results. Overall, our results highlight the importance of considering BMI as a significant variable that influences individual physiological responses to changes in bed inclination.

Abbreviations

ARDS	Acute respiratory distress syndrome
C_{RS}	Respiratory system compliance
EELV	End-expiratory lung volume
$PaCO_2$	Pressure of carbon dioxide
BMI	Body mass index
PaO_2/F_IO_2	PaO_2 over the fraction of inspired oxygen
PEEP	Positive end-expiratory pressure
C_{CW}	Chest wall compliance
VR	Ventilatory ratio
VD_{Bohr}/VT	Bohr dead space fraction
SnIII	The phase III slope of the capnogram (SIII) normalized with fraction of expired CO_2

Supplementary Information

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Supplementary Material 1

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Author contributions

MHB, GB, AB, and JR contributed to the conception and design of the study. All authors performed the title and abstract screening. MHB, RB, TL, FM, JJM, JS, CG, MM, SB, UW, JD, and JR performed the data extraction. MHB, RB, TL, FM, JJM, JS, CG, MM, SB, UW, ELVC, and JR organized the data and created the characteristic tables. MHB and RB performed the data analysis. MHB, TL, FM, JJM, JS, CG, MM, SB, UW, JD, AB, GB, ELVC, JD, and JR wrote the first draft of the manuscript. All authors contributed to manuscript revision, read, and approved the submitted version.

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Data availability

The data sets used and analyzed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate

Not applicable.

Consent for publication

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Competing interests
Dr. Costa received funding from Magnamed and Timpel.Dr. Mehdi Mezidi reports receiving congress fees from Pfizer, unrelated to the present work.The other authors declare that they have no competing interests.

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